Axon cytoskeleton proteins specifically modulate oligodendrocyte growth and differentiation in vitro

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Résumé en anglais	In multiple sclerosis (MS) remyelination by oligodendrocytes (OL) is incomplete, and it is associated with a decrease in axonal neurofilaments (NF) and tubulin (TUB). To determine whether these proteins could participate directly in MS remyelination failure, or indirectly through proteins that are co-associated, we have analysed their effects in pure OL cultures. Rat brain NF fractions, recovered by successive centrifugations increase either OL progenitor (OLP) proliferation (2nd pellet, P2), or only their maturation (P5), whereas albumin, liver and skin proteins, as well as recombinant GFAP or purified actin were ineffective. NF (P2) copurify mainly with TUB, as well as with other proteins, like MAPs, Tau, spectrin β2, and synapsin 2. These purified, or recombinant, proteins increased OLP proliferation without delaying their maturation, and appeared responsible for the proliferation observed with P2 fractions. Among putative signaling pathways mediating these effects Fyn kinase was not involved. Whereas NF did not alter the growth of cultured astrocytes, the NF associated proteins enhanced their proliferation. This suggests that NF and their associated proteins exert specific effects on OL development, broadening the field of axon–oligodendrocyte interactions. In case of axon damage in vivo, extracellular release of such axonal proteins could regulate remyelination and astrocytic gliosis.
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